An Overview of Research Findings on the Nature of Posttraumatic Stress Disorder

PAULA P. SCHNURR
MATTHEW J. FRIEDMAN
Department of Veterans Affairs National Center for PTSD
Dartmouth Medical School

In this article we review current knowledge about the nature of posttraumatic stress disorder (PTSD) in order to assist clinicians in the diagnosis and treatment of reactions to traumatic life events. The review is limited to information about PTSD in adults, although some of the material may generalize to child and adolescent populations. Information presented includes prevalence, course, and comorbidity. We also briefly summarize findings on information processing and psychobiology. After presenting research findings, we then discuss the assessment of trauma and PTSD.

• **PTSD** • **trauma**

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Posttraumatic stress disorder (PTSD) was formally introduced into the psychiatric nomenclature in 1980 (American Psychiatric Association; *DSM-III*). Prior to that time, posttraumatic syndromes were recognized by a variety of names, including DaCosta's syndrome, soldier's heart, railway spine, shell shock, traumatic (war) neurosis, concentration camp syndrome, and rape trauma syndrome. The phenomenology described in these syndromes overlaps considerably with that we would now recognize as PTSD. Only the label is relatively new.

EPIDEMIOLOGY AND PHENOMENOLOGY

Prevalence

There is no universally accepted definition of what constitutes a traumatic event. In general, though, most researchers and clinicians agree that traumatic events involve life threat, and many believe that uncontrollability and unpredictability are important features of a trauma. Specific examples in-

• *Correspondence and requests for reprints should be sent to Paula P. Schnurr, Ph.D., or Matthew J. Friedman, M.D., Ph.D., National Center for PTSD (116D), VA Medical and Regional Office Center, White River Junction, VT 05009.*

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clude war zone exposure, assault, rape, torture, childhood physical and sexual abuse, natural and technological disasters, and serious accidents. Experiencing these events directly, as well as witnessing them or learning that they have happened to a loved one, can be traumatic.

The initial definition of PTSD in *DSM–III* reflected a belief that traumatic events were rare. In fact, traumatic events are a surprisingly common occurrence. Reporting data from the National Comorbidity Survey, a large, nationally representative sample of men and women, Kessler, Sonnega, Bromet, Hughes, and Nelson (1995) indicate that 60% of men and 50% of women experience a traumatic event at some point in their lives, and that the majority of people who have experienced trauma report two or more events. The prevalence of trauma can be even higher in clinical settings, where 80% or more of treatment-seeking individuals may have been traumatized.

Men and women differ in the types of events they are likely to experience. The most common events, overall, are witnessing someone being badly injured or killed, being involved in a fire or natural disaster, and being involved in a life-threatening accident, but all of these are more common in men than in women. Men are also more likely than women to have experienced physical attacks, combat, and being threatened with a weapon, held captive, or kidnapped. In contrast, women are more likely to have experienced rape, sexual molestation, childhood parental neglect, and childhood physical abuse.

General population estimates of lifetime PTSD in the National Comorbidity Survey are 5% for men and 10% for women. Among traumatized individuals, the lifetime prevalence of PTSD is 8% in men and 20% in women. The lifetime prevalence is 31% in male veterans and 27% in female veterans who served in the Vietnam theater of operations, according to the nationally representative sample assessed in the National Vietnam Veterans Readjustment Study (Kulka et al., 1990). The prevalence of PTSD in treatment-seeking populations is much higher, sometimes reported to be in excess of 50%, even among individuals who are not seeking specialized trauma care.

The likelihood of developing PTSD varies with the type of trauma experienced. Such differences between traumas provide a possible explanation for the gender differences in PTSD prevalence, namely, that women are more likely to develop PTSD because of the nature of the traumas they tend to experience. For example, rape, which has the highest likelihood of resulting in PTSD, is much more likely to happen to women (9%) than to men (<1%). However, gender differences in exposure do not completely explain gender differences in prevalence. Women remain 4 times more likely than men to develop PTSD even when type of trauma is taken into account (i.e., controlled for in statistical analysis). Types of trauma for which women are more likely than men to develop PTSD include sexual molestation (27% vs. 12%), physical attack (27% vs. 2%), and physical abuse (49% vs. 22%); rape is the only trauma for which women are substantially less likely than men to develop PTSD (46% vs. 65%). The gender difference in PTSD prevalence requires further explanation, but has important clinical implications nonetheless. Practitioners working with recent trauma survivors may view gender as being associated with increased risk of PTSD and may target their interventions accordingly.

The likelihood of developing PTSD also varies with other risk factors, including both situational and personal characteristics. One important factor is the severity of a trauma, roughly, the "dose." Trauma severity refers to such features as the duration of a trauma, the number of times it occurred, and the extent of perceived in-

jury or life threat. It is our impression that many people mistakenly believe that the relationship between trauma severity and PTSD is much stronger than it actually is. In fact, the magnitude of the relationship between trauma severity and PTSD is only moderate, but it is highly consistent across populations and trauma types: the greater the trauma severity, the greater the likelihood of PTSD or severity of PTSD symptoms.

Age and education level at time of trauma are other risk factors for PTSD. Younger age, both in childhood and early adulthood, and lower education are associated with increased risk. Increased risk of PTSD also is associated with severity of initial reaction, peritraumatic dissociation (i.e., during and shortly following a trauma), early conduct problems, childhood adversity (e.g., parental loss, economic deprivation), family history of psychiatric disorder, poor social support after a trauma, and personality pathology (especially neuroticism).

An additional issue in the epidemiology of PTSD is that the distinction between cases and noncases does not reflect an absence of symptoms among noncases, but rather, a failure to meet all of the diagnostic criteria for the disorder. Posttraumatic reactions fall on a continuum, and although full PTSD is a valid diagnosis, a number of clinicians and researchers believe that it is useful to conceptualize partial syndromes as well. Definitions of partial PTSD typically require that the *B* (reexperiencing) symptom criterion be met along with some combination of the *C* (avoidance/numbing) and *D* (hyperarousal) criteria, for example, meeting *B* and *D* while having fewer than three *C* symptoms. Estimates of partial prevalence from the National Vietnam Veterans Readjustment Study (Kulka et al., 1990) indicate the potential clinical utility of recognizing and treating partial syndromes: The lifetime prevalence of partial PTSD was 23% among men and 21% among women who served in the Vietnam theater.

Course

The Diagnostic and Statistical Manual of Mental Disorders (*DSM–IV;* American Psychiatric Association, 1994) includes several specifiers for providing information about the course of PTSD. An acute case is one in which the duration of symptoms is less than 3 months. A chronic case is one in which symptoms last 3 months or longer. Delayed onset of PTSD is diagnosed when at least 6 months have elapsed between traumatic exposure and the onset of symptoms.

Prospective studies that chart the average number of symptoms or percentage of PTSD cases at different times following a trauma show an average pattern of decline, but averages can provide misleading information about symptom change in individuals. Although it is true that, *on average,* posttraumatic symptoms decrease over time, the course of PTSD is quite variable, both within and between people. The exact percentages of people who have immediate versus delayed onset, a single versus multiple symptomatic episodes, and a variable versus a stable course, are unknown. However, delayed onset does not appear to be the norm, nor does a stable course.

Studies that estimate both lifetime and current prevalence of PTSD often find that roughly half of the people who had PTSD at some point in the past do not meet diagnostic criteria at the time of assessment. This does not mean that half of the people who develop PTSD recover. Individuals who are asymptomatic at one time may find that their symptoms are reactivated by stressors, both related and unrelated to the initial trauma. For some people, the duration of asymptomatic or mildly symp-

tomatic periods can be very long. For example, World War II combat veterans who had not thought about their war experiences in years found themselves having intrusive thoughts, nightmares, and other PTSD symptoms after media broadcasts of various 50th anniversary events of the war. Also, retirement, or serious medical illness in a survivor or his or her spouse, can reactivate symptoms. In general, the increases in PTSD symptoms during late life are not extreme cases of delayed onset, but rather, are cases of symptom recurrence or delayed recognition.

Estimates of the extent of chronicity depend on the duration criterion used to determine PTSD. The National Comorbidity Survey (Kessler et al., 1995) reported that symptoms lasted more than 1 year in over two thirds of PTSD cases, and more than 3 years in over one third of PTSD cases. There is little empirical knowledge about risk factors for chronic PTSD. It is possible that the same factors that are associated with risk of PTSD also increase risk of chronicity. Early intervention, which is believed to decrease the risk of developing PTSD, also might decrease the risk of chronicity, but the existing data do not permit strong conclusions at this time.

Among chronic cases, disabling symptoms may persist with periodic exacerbations throughout the life span. Chronic PTSD is often accompanied by other Axis I psychiatric disorders and general impairment of psychosocial function. We address this and related issues in greater depth in the following section.

Comorbidity

Like many other *DSM–IV* diagnoses, PTSD often co-occurs with at least one other disorder. In the National Comorbidity Survey, Kessler et al. (1995) reported that approximately 80% of all men and women with lifetime PTSD exhibited one or more comorbid diagnoses. With respect to specific comorbid disorders for men and women, the lifetime prevalence was approximately 48% for major depressive disorder (MDD), 22% for dysthymia, 16% for generalized anxiety disorder (GAD), 30% for simple phobia, and 28% for social phobia. Notable gender differences were also found. Women exhibited greater lifetime prevalence of panic disorder and agoraphobia, whereas men exhibited greater lifetime prevalence of alcohol abuse/dependence, drug abuse/dependence, and conduct disorder.

Comorbid lifetime substance abuse is a relatively common clinical occurrence that complicates treatment considerably. General population estimates for comorbid alcohol abuse/dependence are 52% for men and 28% for women. The respective prevalences for comorbid drug abuse/dependence are 35% and 27%. In the majority of such cases, PTSD is the primary disorder.

Certainly one reason for the high lifetime comorbidity seen in PTSD is the considerable overlap between symptoms associated with PTSD and several other psychiatric disorders. For example, both PTSD and MDD share insomnia, impaired concentration, and diminished interest in one's surroundings. In addition, emotional detachment and restricted range of affect in PTSD may be confused with depressed mood and psychomotor retardation in MDD. Furthermore, (survivor) guilt and suicidal behavior, which are symptoms of MDD, are often seen in individuals with PTSD, although these symptoms are not included among the *DSM–IV* diagnostic criteria. Symptoms found in both PTSD and GAD include: autonomic hyperarousal, irritability, hypervigilance, exaggerated startle, impaired concentration, and insomnia. Finally, PTSD and panic disorder both exhibit autonomic hyperarousal and dissociation but, perhaps more important, the psychological and phys-

iological reactivity in PTSD can sometimes appear to be a full-fledged panic attack.

The prominence of comorbidity and symptom overlap between PTSD, MDD, and other anxiety disorders raises important theoretical and clinical issues. Theoretically, one may ask whether a person who meets diagnostic criteria for PTSD and MDD or PTSD and GAD has two distinct disorders or whether these really represent depressive or anxious subtypes of PTSD itself. Indeed, there is compelling neurobiological evidence suggesting that comorbid PTSD and MDD is much more similar to PTSD alone and distinctly different from MDD alone. The evidence for this speculation will be presented subsequently.

Clinically, comorbidity and symptom overlap demand that all new psychiatric patients receive a thorough diagnostic assessment, including a comprehensive trauma history. Unfortunately this is not always the case. As a result, an individual with severe PTSD who presents as an acute emergency with suicidal behavior, autonomic hyperarousal, or alcohol/drug intoxication may be diagnosed and treated exclusively for MDD or panic disorder or chemical abuse/dependency. It is not unusual to discover afterwards that the acute psychiatric emergency was due primarily to the underlying PTSD. Failure to conduct a comprehensive assessment in such cases may result in poor clinical management and sometimes in tragic consequences.

It is important to consider medical as well as psychiatric comorbidity among individuals with PTSD. There is mounting evidence to suggest that trauma is a risk factor for poor physical health. Trauma survivors, relative to nontraumatized individuals, report more medical symptoms, use more medical services, have more medical illnesses confirmed by a physician's examination, and display higher mortality. A few studies suggest that PTSD plays an important mediational role in the relationship between trauma and physical health. This is an attractive hypothesis, as we have found that correlates of PTSD—neurobiological (including immunologic) abnormalities and psychological factors (depression, hostility, negative health behaviors, and poor coping abilities)—are associated with increased risk of medical illness (Friedman & Schnurr, 1995). One implication of this finding on trauma and adverse health outcomes is that trauma histories and PTSD assessment should become a routine component of all diagnostic evaluations by primary and specialty medical practitioners.

Complex PTSD

Some clinicians and researchers have argued that although the PTSD diagnostic criteria adequately encompass the symptoms experienced by most people who are exposed to catastrophic stress, these criteria do not include clinically significant symptoms exhibited by some people who have been exposed to protracted interpersonal trauma such as childhood sexual abuse, domestic violence, and political torture. Even though these individuals usually meet diagnostic criteria for PTSD, it has been suggested that there is a different, comorbid constellation of symptoms, called "complex PTSD," that is much more pertinent. As defined by Herman (1992), people who have complex PTSD are characterized by problems with impulsivity, affect regulation, dissociative symptoms, self-destructive behavior, abnormalities in sexual expression, and somatic symptoms.

It is presently controversial whether complex PTSD should become a separate diagnostic entity in its own right. There is general agreement, however, that individuals who exhibit this pattern of symptoms often present a very difficult clinical challenge. The first priority of treatment is to help them acquire a safe and stable

emotional and physical environment by eliminating risky behaviors, destructive addictions, abusive relationships, and dangerous living situations. It is only after such stabilization has been achieved that treatment can focus on the traumatic event itself. In the third and final phase, treatment addresses behavioral and affect management in a here-and-now context with emphasis on interpersonal, marital, family, vocational, and social functioning.

Ethnocultural Considerations

It must be emphasized that the epidemiological and comorbidity data presented previously only apply to studies of American men and women. Such information is not currently available from other nations. It seems likely, however, that PTSD prevalence will be much higher in nations where exposure to war, state-sponsored terrorism, or interpersonal violence is much higher than in the U.S. This is particularly applicable to nations in which genocide and other crimes against humanity have recently been perpetrated on a massive scale, such as Rwanda, Bosnia, and Cambodia.

It must also be emphasized that the diagnostic criteria for PTSD and most of the current data pertaining to this disorder are based primarily on research and clinical experience with North American or European individuals. Indeed, PTSD has been criticized from a cross-cultural perspective because of this Western, industrialized, Euro-American bias. The question is not whether PTSD can be detected among traumatized people from traditional ethnocultural settings (e.g., Southeast Asians, Latin Americans, Africans), because it certainly has been. Rather, the question is whether there may be other distinctive ethnocultural posttraumatic idioms of distress that fall outside strict *DSM–IV* guidelines, but that are major indicators of significant posttraumatic distress in their own right. Marsella, Friedman, Gerrity, and Scurfield (1996) noted that it is important to understand how traumatic exposure is perceived and expressed in different ethnocultural settings, because different clinical interventions may be required than those that have proven effective for Euro-American individuals with PTSD.

INFORMATION PROCESSING IN PTSD

Memory and concentration difficulties are frequent complaints among people who have PTSD. Findings regarding the exact nature of cognitive problems in PTSD are inconsistent—for example, some data show the deficits to be fairly specific, whereas other data show the deficits to be more general—but one point is clear: The subjective complaints of cognitive problems are corroborated by poor performance in assessments of cognitive function. An exciting area of PTSD research is focused on answering questions about the extent to which these cognitive problems in PTSD are related to alterations of brain structures involved in learning and memory.

A widely replicated laboratory finding is that individuals with PTSD selectively attend to and process trauma-relevant information. One method used to investigate selective processing is the Stroop color-naming test, in which the dependent measure is the latency to name the color in which a word is printed. Relative delays in color-naming latency are taken as an indicator that the word's meaning has interfered with task processing (e.g., the word "blue" is more quickly identified when

printed in blue than in red). Investigators using this paradigm have suggested that it may be an indicator of the symptom of intrusion. Individuals with PTSD are more likely than those without PTSD to show greater interference when presented with trauma-related words. Most data show that PTSD is not associated with a more generalized differential responsiveness to threatening words that are unrelated to trauma. Additional studies have shown that individuals with PTSD show biases favoring trauma-related material in tasks designed to tap explicit memory (e.g., recall, recognition) as well as implicit memory (e.g., word-stem completion, lexical decision).

Several authors have proposed information processing models of PTSD. For example, Foa, Steketee, and Rothbaum (1989) offered a model based upon the concept of a "fear structure," which they described as a "network in memory that includes three types of information: (a) information about the feared stimulus situation; (b) information about verbal, physiological, and overt behavioral responses; and (c) interpretive information about the meaning of the stimulus and response elements of the structure" (p. 166). Fear structures are programs that enable individuals to escape or avoid a feared stimulus. They differ from other information structures in memory not only because of their particular stimulus and response components, but also because fear structures contain information about danger. Foa and her colleagues have proposed that treatment must be based upon the activation and correction of information in fear structures, accomplished by exposure to traumatic stimuli and cognitive restructuring, respectively (see Jaycox, Zoeller, & Foa, in this issue). The model has yielded much productive research and has served as the basis for a theoretically grounded approach to treatment.

PSYCHOBIOLOGY OF PTSD

There is a rich tradition of research on stress, coping, and adaptation that serves as a useful context in which to understand many of the psychobiological abnormalities detected among individuals who have PTSD. A number of key biobehavioral systems have been selected through evolution to promote survival of the human species. These include sympathetic nervous system (SNS) activation, mobilization of the hypothalamic–pituitary–adrenocortical (HPA) axis, fear conditioning, and the startle reflex. A thorough review of animal and human research in this area can be found in Friedman, Charney, and Deutch (1995).

The discovery of the crucial role of SNS activation as a survival mechanism is generally attributed to Walter B. Cannon (1932), who called it the "fight or flight response." This is a complex, but efficiently orchestrated, response by mammals to threatening situations in which central nervous system (CNS), SNS, and neuromuscular mechanisms are rapidly recruited by the adrenergic nervous system to promote survival. Normally, when the crisis has passed and coping has been successful, adrenergic activity returns to precrisis normal homeostatic levels. That is not so for people with PTSD, who exhibit continuously elevated SNS and adrenergic function, even in the absence of an external threat. For example, people who have PTSD exhibit SNS hyperresponsivity to a variety of neutral and trauma-related stimuli presented in a laboratory. Elevated concentrations of adrenergic metabolites (catecholamines) are found in their urine. Some (alpha-2 and beta) adrenergic receptors are down-regulated and are unusually sensitive to certain

drugs (e.g., yohimbine) that disinhibit the CNS adrenergic system. In short, the adrenergic system in people who have PTSD appears to have been recalibrated to deal with a permanent, life-threatening crisis.

The HPA system is the other key biobehavioral system involved in the human response to stress. Here, too, there is elegant evidence showing continuous dysregulation of HPA function among individuals with PTSD, who exhibit lower urinary cortisol levels, elevated lymphocyte glucocorticoid receptor levels, and supersuppression to the glucocorticoid dexamethasone during the dexamethasone suppression test (DST). Research on the DST is extremely important, not only because it is the most elegant demonstration of psychobiological dysregulation associated with PTSD, but because it may have an important clinical application as a psychobiological assessment tool. Yehuda (1995) has found that, whereas men and women with neither PTSD nor MDD exhibit HPA suppression during the DST, those with MDD are nonsuppressors and those with PTSD are supersuppressors who exhibit HPA suppression at one quarter or one half the dose needed to produce HPA suppression in normal individuals. Furthermore, people who have PTSD with comorbid MDD are also predominantly supersuppressors. As noted in our discussion of comorbidity, this suggests that PTSD with comorbid MDD may actually be a depressive subtype of PTSD rather than two distinct psychiatric disorders as diagnosed by the *DSM–IV*.

Fear conditioning is an adaptive mechanism whereby animals learn to preserve information about previous threats in order to promote future survival. The most elegant research demonstrating the presence of fear conditioning among individuals with PTSD involves laboratory paradigms in which these individuals are exposed to auditory or visual stimuli pertaining to their traumatic event. For rape victims this might be stimuli about sexual assault, for military veterans it might include war zone related stimuli, and for both it might include a brief audiotaped, autobiographical synopsis of their specific traumatic episode. Most people who have PTSD will exhibit sudden dramatic elevations of cardiovascular or other SNS activity immediately after exposure to such trauma-related stimuli in a laboratory setting.

The startle response reflects the organism's alarm system upon exposure to threatening stimuli. Abram Kardiner's (1941) observations of exaggerated startle among World War I veterans suggested to him that PTSD (i.e., "shell shock," "traumatic neurosis") was an abnormal physiological as well as psychological state. As a result, he called this condition a "physioneurosis." Abnormal findings in startle reflex research with people who have PTSD include shorter latency and increased amplitude, resistance to normal habituation, and loss of the normal inhibitory modulation of the startle reflex.

Well-established abnormalities detected in other major biobehavioral systems among individuals with PTSD include alterations in the diurnal sleep cycle, elevated thyroid function, and dysregulation of the opioid system. Less well-established, but strongly supported by relevant animal or human research, is the likelihood that PTSD may be associated with suppressed immunologic function, kindling/behavioral sensitization of specific brain nuclei (especially in the hippocampus and other limbic system sites), abnormal serotonergic activity, and altered glutamatergic (excitatory amino acid) mechanisms that might adversely affect information processing and memory function.

Given the wide spectrum of psychobiological abnormalities detected among individuals with PTSD, it might be expected that they would also exhibit abnormal-

ities in brain structure and function. Although research on this possibility is in its infancy, several independent laboratories have reported that such abnormalities are present. Three independent studies using magnetic resonance imaging have shown reduced hippocampal volume among men and women with PTSD who have been exposed to sexual, war zone, and motor vehicle trauma, respectively. Functional brain abnormalities are suggested by two investigations with positron emission tomography (PET) in which individuals with PTSD who are exposed to trauma-related stimuli exhibited increased regional cerebral blood flow to brain structures (e.g., limbic and paralimbic areas, especially the amygdala and anterior cingulate gyrus) that appear to play key roles in processing emotional information. The PET findings are completely consistent with what one would expect if brain mechanisms underlying the stress ("fight or flight") response or fear conditioning are dysregulated in PTSD.

ASSESSMENT AND DIAGNOSIS

This section provides information about the assessment of PTSD. Our assumption is that readers of this journal are primarily interested in assessment for clinical purposes, and we have tailored our comments accordingly. With one exception, we do not mention specific instruments. Instead, we present general principles and encourage readers to examine the recent volume by Wilson and Keane (1996) for more specific information about instruments as well as comprehensive discussion of many broader issues.

Assessment of Trauma

The first step in the diagnosis of PTSD is determining whether or not an individual has been exposed to a traumatic event, or, as will often be the case, determining to which events an individual has been exposed. This step is important because exposure is one of the diagnostic criteria for PTSD and because all of the *B* (reexperiencing) symptoms and two *C* symptoms must be evaluated with respect to a specific event or events. The decision to query about a single event or to obtain a lifetime trauma history (and then perhaps focus on a few key events) should depend on whether you need to know if an individual has PTSD in response to a specific event only, for example, whether a childhood sexual trauma survivor who has been in a motor vehicle accident meets criteria for PTSD related to the accident. If not, you may elect to assess PTSD in response to a worst event, a most recent event, or a constellation of a few of the worst events.

The first diagnostic criterion for PTSD, as listed in *DSM–IV,* requires that an individual has been exposed to (or witnessed or has been confronted with) a life-threatening event to which the individual responded with "fear, helplessness or horror." Clinical judgment may be required to determine whether an event was life threatening. Individuals sometimes exaggerate, but also sometimes minimize, the life threat in a given situation. Assessing whether serious injury or death occurred for someone involved in the situation can be very useful for ascertaining life threat, although many traumatic events do not result in actual injury or death to anyone. Some suggested probes are: "Were you or others in the situation seriously injured or harmed? [IF NO, OR NOT SURE] Did you think that your life was in danger or that you might be seriously injured? Did you think that others might be killed

or seriously injured? Under such circumstances, do you think that other people would feel that their lives were in danger or that they might be seriously injured?"

Clinical judgment also is required to determine an individual's response to a trauma. Again, exaggeration may be a problem, but it is not uncommon for people to report being numb, or "in shock." In this case, asking about an individual's reaction at some point after the trauma—"when the shock wore off"—can be useful.

Our mention of the importance of clinical judgment raises the question of whether it is better to use interviews or questionnaires for assessing trauma. There is no absolute answer to this question. Obviously, interview assessments can be essential for dealing with exaggeration and minimization, but interviews may be subject to problems of interrater reliability as well as the unwillingness of some people to disclose to another person what they might disclose in an anonymous questionnaire. Questionnaires can provide a more comfortable format than interviews to relate distressing experiences, but care must be taken during the administration of questionnaires to ensure that appropriate clinical contact is available if needed.

The small amount of information available comparing questionnaire and interview assessment, though, is that questionnaires may slightly underestimate the amount of exposure, probably because interviews can provide clarification about the meaning of a given item. This may be especially important in the assessment of sexual trauma because individuals may be unsure about categorizing some sexual experiences they have had (e.g., date rape). One practice commonly employed in many trauma questionnaires and interviews is for questions to be behaviorally specific, that is, defined in terms of the behaviors involved ("inserted a penis, finger, or other object into your vagina") rather than summary labels (e.g., "vaginal rape").

Research on the stability of various trauma assessment measures over time indicates that reports of event exposure are reasonably, but not perfectly, consistent from one occasion to another. This research has not conclusively determined why individuals may report an event on one occasion and fail to report it on another, or vice versa. Simple forgetting is a likely explanation for some inconsistency, as is reluctance to retell a given event to a given person on a given day. There is a widely held clinical impression that either traumatic exposure or PTSD itself may interfere with the retrieval of traumatic memories in some individuals. This issue is currently generating important research on the nature of memory processes in general, and traumatic memories in particular.

A related issue is the validity of a report of trauma on a single occasion. During the past few years, the question of how to determine the validity of a traumatic memory has provoked a great deal of controversy, with claims in both forensic and clinical settings that memories of traumatic events can be falsely implanted by inappropriate clinical practices. Thorough discussion of this complex issue is beyond the scope of this article, but we note that there seems to be reasonable consensus that suggestive memory-recovery techniques should be used with extreme caution in clinical interactions with individuals who have unclear memories, or no memories at all. For further information, readers should consult a special issue of the *Journal of Traumatic Stress* (Green & Wolfe, 1995) that was devoted to the topic of traumatic memory and to the assessment book by Wilson and Keane (1996).

Some researchers have attempted to examine the validity of individuals' reports of exposure to traumatic events by comparing these reports with archival sources such as combat medals or state records of child abuse. The comparisons have shown that self-reports and archival records are reasonably correlated, but that individuals may sometimes fail to report documented trauma. An important problem with

archival sources is that they are not a complete record of an individual's experiences and may underestimate exposure. Documented occurrences may be very good indicators of exposure, but lack of documentation does not necessarily indicate that exposure never took place. It is helpful to seek out archival and collateral sources of information when possible (and appropriate), but these sources should be used in conjunction with an individual's own report to develop a comprehensive picture of his or her trauma history.

Assessment of PTSD

The current diagnostic criteria for PTSD categorize symptoms into three clusters: (a) *persistent reexperiencing* of the traumatic event; (b) *avoidance* of stimuli associated with a traumatic event or *numbing* of general responsiveness (not present before the event); and (c) *increased arousal.* These symptoms must last for at least 1 month and must result in either significant distress or psychosocial impairment. PTSD is distinctive among psychiatric disorders in that its diagnosis requires the identification of an etiological factor, which presents a challenge for both the traumatized individual and the assessor to determine explicitly to what extent the individual's symptoms are a *reaction* to a trauma. This task is straightforward for reexperiencing and avoidance symptoms, which are literally referenced to the trauma, but less so for numbing and hyperarousal. For these symptoms it is necessary to determine whether their onset occurred after the trauma, or if not, whether their severity intensified after the trauma. It may not be possible to ascertain the temporal relationships between trauma exposure and trauma-nonspecific symptoms in an individual who was traumatized at a young age. In such cases, the symptom may be considered to be trauma-related.

At times, obtaining a lifetime trauma history can lead to complications for the clinician or researcher, who, upon finding out that a traumatized individual has experienced more than one event, must confront the question of what event or events to use as the basis for PTSD assessment. There is no simple answer to this question. It is uncommon to assess whether an individual has PTSD in response to every event that has been experienced. A more typical practice is to assess PTSD with respect to a "worst" event, a first event, a most recent event, or some manageable combination of events simultaneously. The latter can be particularly useful because individuals who have experienced multiple traumas often will have a blended mapping of traumas to symptoms (e.g., nightmares about combat but intrusive recollections about combat and a serious auto accident).

Questionnaire measures of PTSD fall into three general categories: (a) items that represent the symptoms needed for diagnosis; (b) items needed for diagnosis as well as associated features such as guilt; and (c) items developed empirically from items taken from general symptom inventories. The first two types of questionnaires can be used to determine whether an individual has the sufficient number and type of symptoms needed for diagnosis. All questionnaires can be used to determine whether an individual is likely to have a diagnosis based on an overall severity score. For some questionnaires in particular, this severity threshold approach is a highly valid way to estimate a diagnosis. Besides their obvious value in assessing a large number of people in a cost-efficient manner, questionnaires are useful for screening and for tracking symptom severity.

However, structured interview measures of PTSD are considered to be the gold standard for diagnosis. Although clinical interviews are often used by busy practitioners, the additional time that can be required to administer a more structured in-

terview is justified by the completeness and accuracy such methods foster. Available interviews vary in terms of whether they require a clinician or a layperson for administration, provide severity information along with a diagnosis, and include questions about traumatic exposure and associated symptom features. Layperson-administered interviews are typically used in research settings, either for screening or when budget constraints preclude the participation of clinicians. These interviews can have acceptable to very good validity when lay interviewers use instruments designed for nonclinicians, and are carefully trained and monitored.

There are a number of excellent structured interviews available for clinically trained researchers and practitioners. Here we describe one of them in detail, the Clinician-Administered PTSD Scale, or CAPS. The CAPS was developed by Blake et al. (1995) at the National Center for PTSD as an interview for diagnosing current and lifetime PTSD, and for quantifying PTSD symptom severity. It has two versions: the DX, for diagnosis, and the SX, for assessing weekly change. Both versions can provide a diagnosis and a severity score, but the SX is primarily used for the latter purpose. The CAPS consists of questions about the 17 PTSD symptoms listed in *DSM–IV*, duration, course, impairment, and associated symptoms, including guilt, suicidality, and dissociation. It also includes a simple and clinically helpful trauma assessment procedure for determining the event(s) to be used as the basis for PTSD assessment.

Several aspects of the CAPS make it an informative and easy-to-administer interview. For each symptom, respondents are asked about the symptom's frequency, which is rated on a 5-point scale. Then, provided that the symptom occurs, respondents are asked about its intensity, which is also rated on a 5-point scale. Ratings of frequency and intensity are highly correlated. However, distinguishing them can be important for getting a good picture of intense but low-frequency events as well as for tracking symptom change, which may occur by decreases in frequency or intensity, but not both.

Sample CAPS items are illustrated in Figure 1, which highlights another useful feature of the CAPS: behavioral descriptions to help the interviewer distinguish between each point on the frequency and intensity scales. A "questionable validity" (or QV) specifier is included for indicating concerns about a rating's quality (e.g., vague or inconsistent examples or poor comprehension on the part of a respondent). A "trauma-related" specifier is additionally included on items that are not specifically referenced to the trauma (e.g., restricted range of affect). This specifier permits an interviewer to acknowledge a symptom's presence while discounting it for diagnostic purposes.

The severity of an individual symptom is computed by summing intensity and frequency ratings. A total severity score is computed by then summing the individual symptom severity ratings. The presence or absence of each symptom may be determined according to several rules. The most widely used rule is to count a symptom as present if it is at least a "1" in frequency and a "2" in intensity, that is, present and of at least moderate intensity. Research indicates that this rule probably is too liberal, leading to the overdiagnosis of PTSD, and that the optimal severity (frequency + intensity) score for making a diagnosis varies from item to item. One study found the cutoff for each of the 17 *DSM–IV* items to be, in order: *2* for Item 2; *3* for Items 1, 3–5, 16, and 17; *4* for Items 6, 7, and 13–15; *5* for Items 8–10 and 12; and *6* for Item 11. In general, the "1–2" rule is useful for clinical intake purposes, but a more stringent scoring criterion should be considered when diagnostic validity is an important concern, such as in a forensic context.

Criterion B. The traumatic event is persistently reexperienced in one (or more) of the following ways:

1. (B-1) recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions.
Note: In young children, repetitive play may occur in which themes or aspects of the trauma are expressed.

Frequency	***Intensity***	***Current***
Have you ever had unwanted memories of (EVENT)? What were they like? *(What did you remember?)* [IF NOT CLEAR:] *(Did they ever occur while you were awake, or only in dreams?)* [EXCLUDE IF MEMORIES OCCURRED ONLY DURING DREAMS] **How often have you had these memories in the past month?** 0 Never 1 Once or twice 2 Once or twice a week 3 Several times a week 4 Daily or almost every day ***Description/Examples***	**How much distress or discomfort did these memories cause you? Were you able to put them out of your mind and think about something else?** *(How hard did you have to try?)* **How much did they interfere with your life?** 0 None 1 Mild, minimal distress or disruption of activities 2 Moderate, distress clearly present but still manageable, some disruption of activities 3 Severe, considerable distress, difficulty dismissing memories, marked disruption of activities 4 Extreme, incapacitating distress, cannot dismiss memories, unable to continue activities ***QV (specify)*** _____	***F*** _____ ***I*** _____ ***Sx: Y N*** ***Lifetime*** ***F*** _____ ***I*** _____ ***Sx: Y N***

Figure 1. Question example from the Clinician-Administered PTSD Scale (CAPS; Blake et al., 1995).

Multimodal assessment of PTSD is recommended. Combining questionnaire and interview data, and, if possible, psychophysiological and neurobiological data, provides the most comprehensive means of making a diagnosis and characterizing an individual's posttraumatic reactions and clinical needs. Unless you only wish to determine whether a given individual has PTSD, it is desirable to assess other symptoms as well as psychosocial impairment. As noted in our discussion of comorbidity, chronic PTSD is often associated with a complex array of problems that can affect many domains of functioning.

CONCLUDING REMARKS

PTSD is a disorder with a presumed etiology: a traumatic event. Yet most people who experience a trauma never develop PTSD. A traumatic event appears to be necessary, but not sufficient, for producing PTSD. The relatively low prevalence of PTSD among individuals who are traumatized raises the question of what factors besides a trauma are required in order for PTSD to develop. Studies published in the early and mid-1980s tended to show little, if any, contribution of premorbid characteristics such as childhood environment. Some findings pointed to the im-

portance of social support following a trauma as a predictor, but this early research consistently identified trauma severity as the primary etiological agent. After almost two decades of study, a large amount of data suggests that the etiology of PTSD is best understood in terms of what an individual brings to a traumatic event as well as what he or she experiences afterward, and not just characteristics of the event itself. Our brief review of the epidemiology of PTSD mentioned several factors: age, education, gender, personality, and social support. Some of the most promising PTSD research has attempted to delineate the relationships among these etiological factors, or has been designed to rule out the possibility of retrospective bias in posttraumatic self-reports of premorbid conditions. A valuable result of this etiological research has been increased understanding of the role of risk factors such as gender in the etiology of trauma exposure itself. Still, prospective studies are much needed, especially those that measure at-risk individuals prior to traumatic exposure and those that frequently assess recently traumatized individuals in order to chart the course of acute posttraumatic reactions.

Abram Kardiner was absolutely correct in 1941 when he called PTSD a "physioneurosis." Neither he nor the rest of us, however, could have possibly anticipated the wide spectrum and severity of psychobiological abnormalities associated with this disorder. It is hoped that our growing understanding of the biobehavioral complexity of PTSD will help us develop more effective treatment for this potentially chronic and disabling disorder.

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